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ABSTRACT

This theme issue addresses environmental risks to infants and young children. Articles have the following titles and authors: "Lead Exposure: The Commonest Environmental Disease of Childhood" (Herbert L. Needleman); "Research in Process: A Comparison of Lead Screening Methods in Young Children" (Catherine S. Rude and David Schonfeld); "Childhood Burns: The Preventable Epidemic" (Jean L. Athey and Laura Kavanagh); "Infants, Toddlers, and Indoor Air Pollution" (Nancy Thorndike Greenspan); "Environmental Risks to the Infants and Toddlers of Migrant Farmworkers" (Jan Greenberg). References accompany each article. (DB)

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Register now for NCCIP's National Training Institute December 6-8, 1991
See page 26

Lead Exposure: The Commonest Environmental Disease of Childhood

by

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Childhood lead poisoning is the most common pediatric environmental disease in the United States. Recent progress in the epidemiology and toxicology of lead exposure has advanced the state of our knowledge base substantially, and set the stage for the complete eradication of this disease.

The awareness of practicing pediatricians, however, has lagged well behind the newer scientific information. Many other professionals are also unaware of the relevance of lead exposure to the development of children. Most evaluation centers for children with learning disabilities or retardation overlook lead as a causative or contributing factor in the deficits displayed by their patients. Many developmental psychologists and other child development professionals use the word "environment" to refer to the social milieu alone, and give no attention to the chemical environment of the brain. These are jarring omissions. The epidemiology of plumbism (lead poisoning), its recognition, and its management are given only minimal attention in many pediatric training programs. A number of excellent pediatric centers have stopped screening for lead exposure. As a result, many otherwise well-trained pediatricians begin practice never having seen a case of lead poisoning. Lead is only rarely considered in the differential diagnosis of mental retardation, developmental delay, speech disorder, behavior deficit, or growth failure.

Ironically, lead may be the first recognized neurotoxin. Two thousand years ago, Dioscerides, a Greek physician, remarked that "Lead makes the mind give way." Two hundred years ago, Benjamin Franklin, in a letter to a friend, clearly described the "dry gripes (colic) and "dangles" (wrist drop) found in tinkers,

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typesetters, and painters (1). Almost one hundred years ago, childhood lead poisoning was first described at the Brisbane Children's Hospital in Australia by A.J. Turner (2) and J.L. Gibson (3). Using painstaking epidemiology they established that the cause was white paint on the porches and rails of the children's homes. After a long and somewhat acrimonious struggle, Turner and Gibson succeeded in 1920 in passing a law forbidding

Contents:

Lead Exposure p.1
Lead Screening Methods in Young Children p.7
Childhood Burns: The Preventable Epidemic p.8
Infants, Toddlers, and Indoor Air Pollution p.14
NCCIP Report, 1990 ..... insert
Environmental Risks to the Infants and Toddlers of Migrant Farmworkers p.22
The Case for a National Infant Care Leave Policy p.24
National Center Notes p.25
Publications p.27
Conference Call p.29
Topical Index for Volume XI p.30

EC 300634

the use of lead paint in dwellings. This preceded its counterpart in American legislation by more than 50 years.

For many years, it was believed that if a child did not die of toxicity, there were no lasting sequelae. In 1943, Randolph Byers showed that children who had recovered from plumbism had a high frequency of learning disorders and behavior problems (4). This paper marked the beginning of the modern era of lead studies.

Lead levels are measured in micrograms per deciliter (about a fifth of a pint) of blood (ug/dl). In the 1970's, the defined toxic level of lead in the blood was 60 ug/dl. Screening studies showed that in some areas as many as 10-20% of children had blood lead levels in the range of 30-40 ug/dl. The question of whether these burdens, in the absence of symptoms of intoxication, were associated with central nervous system damage came under scrutiny. Early studies were in the main crude, and produced conflicting judgments; some found IQ deficits and others did not. This question became highly charged and surrounded with controversy, as proprietary interests inserted themselves into the scientific dialogue. The issue has been effectively settled in recent years, as new studies of quality have been conducted in the U.S., Europe, Scandinavia, Australia and New Zealand.

These later reports show clear lead effects on central nervous system (CNS) function at low doses. The no-effect level has not been found. In addition to CNS function, lead has been found to affect a host of other physiological targets as well, including hearing, blood pressure, and physical growth. Lead toxicity is also associated with anemia.

As a consequence of this new information, the current definition of excess lead exposure, set at 25 ug/dl by the Centers for Disease Control in 1983, will soon be revised downward. Policy statements by the Environmental Protection Agency (EPA) and the Agency for Toxic Substances and Disease Registry (ATSDR) of the Public Health Service (PHS) have asserted that lead-induced neurotoxicity begins at 10-15 ug/dl (5,6).

Low level lead exposure: recent data and continuing research

Knowledge of lead's effects on children's brains has grown exponentially in recent years. Early studies of this question tended to use small sample sizes, coarse measures of outcome, and limited control of covariates (other variables, in addition to the variable of interest that must be controlled in analyses to separate the effects of each variable.) These studies relied on concentrations of lead in the blood to classify exposure. But lead has a rather short residence time in blood and may return to normal in exposed subjects.

For that reason, my colleagues and I used shed baby teeth to identify lead. In 1979, we showed that children with elevated amounts of lead in their teeth, but no signs of toxicity, had lower IQ scores, poorer speech and language performance, and impaired attention (7). Teachers' rating scales of children's classroom perfor-

Correction:

The names of the authors of "Implementing a Multi-site, Multidisciplinary Clinical Trial: The Infant Health and Development Program" (*Zero to Three*, April, 1991) should be spelled as follows: Ruth T. Gross and Christine Haynes.

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mance showed that children with higher exposure to lead showed greater impairment. This study also measured and adjusted for 39 covariates. Similar data were subsequently reported by Yule and his colleagues (8), who observed British schoolchildren. In recent years, studies using larger sample sizes and sophisticated multivariate analysis have shown lead effects on IQ on exposures as low as 10 ug/dl (9,10,11,10). Low level lead exposure is now recognized as a worldwide problem.

Follow-up of our subjects from the 1979 study has shown that the consequences of early exposure endure, and cause enduring problems. In fifth-grade children, earlier exposure to lead was associated with lower IQ, more need for special services, and more grade retention (13). When we followed these subjects into young adulthood, we found that having high lead in one's teeth carried a seven-fold increase in the risk of nongraduation from high school and a six-fold increase in the risk of reading disability. Lead exposure was also associated with lower class rank in the final year of high school, more absenteeism, poorer hand-eye coordination, slower reaction time, and poorer vocabulary scores (14). (Figures 2, 3).

Most reviews of studies of lead and children's IQ are narrative summaries that treat each paper in isolation. Following up on Joel Schwartz' original meta-analysis, we extended the review to 12 informative reports (15). Our meta-analysis shows that the combined inference of all studies is that the effect of lead on IQ scores is real.

Lead crosses the placenta. Prospective studies from birth onward have shown that umbilical cord blood lead levels above 10 ug/dl are associated with decrements in IQ scores as late as 24 months of age(16,17,18). We have followed a sample of 249 children from birth to 57 months of age and find that at the last examination, at 57 months, the effects of in utero exposure is somewhat attenuated. However, the IQ score at 57 months is significantly and inversely related to blood lead levels at 24 months (19). This finding suggests that at 57 months, the effect of *postnatal* exposure has become dominant, although the prenatal effect continues to be statistically significant in the children of low socioeconomic status who also have elevated postnatal exposure to lead.

Most studies of lead exposure have focused on IQ. There is no good reason for this; more critical changes in the brain can be expressed in other behaviors. It is useful to speculate about the real social costs of lead exposure. Lead is associated with increased risk for Attention Deficit Disorder and hyperactivity. Attention deficits and hyperactivity (ADDH), in turn, are risk factors for anti-social behavior. The attributable risk for anti social behavior, given ADDH, is .58—in other words, if a male child has hyperactivity and one sign of conduct disorder, he has a 60 percent chance of appearing on a police blotter multiple times before the age of 18. It may be that 20 percent of delinquency is lead-associated. This is a relationship that we are currently studying.

Editors' note:

We have very mixed feelings as we bring you this issue of *Zero to Three*.

It was an observation by Geraldine O'Brien, Executive Director of the East Coast Migrant Head Start Program, that persuaded us of the importance of addressing environmental hazards in *Zero to Three*. We soon discovered that the Select Committee on Children, Youth and Families of the U.S. House of Representatives had held hearings in September, 1990 on the risks of environmental toxins to children. Herbert L. Needleman and Nancy Thorndike Greenspan, witnesses at that hearing, graciously agreed to contribute articles to *Zero to Three*. As we learned more about the U.S. Maternal and Child Health Bureau's growing involvement in injury prevention, it seemed to us that an article on infants, toddlers, and burns belonged in an issue on environmental hazards—one of the reasons that infants and toddlers suffer so many burn injuries and deaths is that they cannot escape from the "environments" of a burning home or a scalding bath. Just as the rest of us cannot escape, or protect ourselves easily, from toxins in our homes, workplaces, and communities.

Talking with the individual contributors to this issue about their research, intervention, and advocacy efforts was exciting. Reading their manuscripts all together, however, aroused other feelings—among them dread, helplessness, and rage. The part of us that really doesn't want to know about dangers lurking in tap water and living room air *is* reassured by the findings of meticulous research—our dread becomes less "nameless"; we can begin to think about interventions to address an identified problem. But then the rage kicks in—we feel betrayed by The Government, controlled by The Interests.

Fortunately, the articles in this issue of *Zero to Three* provide us with many examples of individual intelligence, integrity and determination, all put to use on behalf of children and families. And perhaps even more important, we are provided with the names, addresses and telephone numbers of committees, alliances, coalitions—the quintessentially American approach to identifying and addressing unmet communal needs.

In a recent speech on burn prevention, Jean Athey of the U.S. Maternal and Child Health Bureau asked a question that seemed an appropriate close to these notes:

"We have to keep on working for our children and not give in to despair ourselves. For if *we* despair, who will fight?"

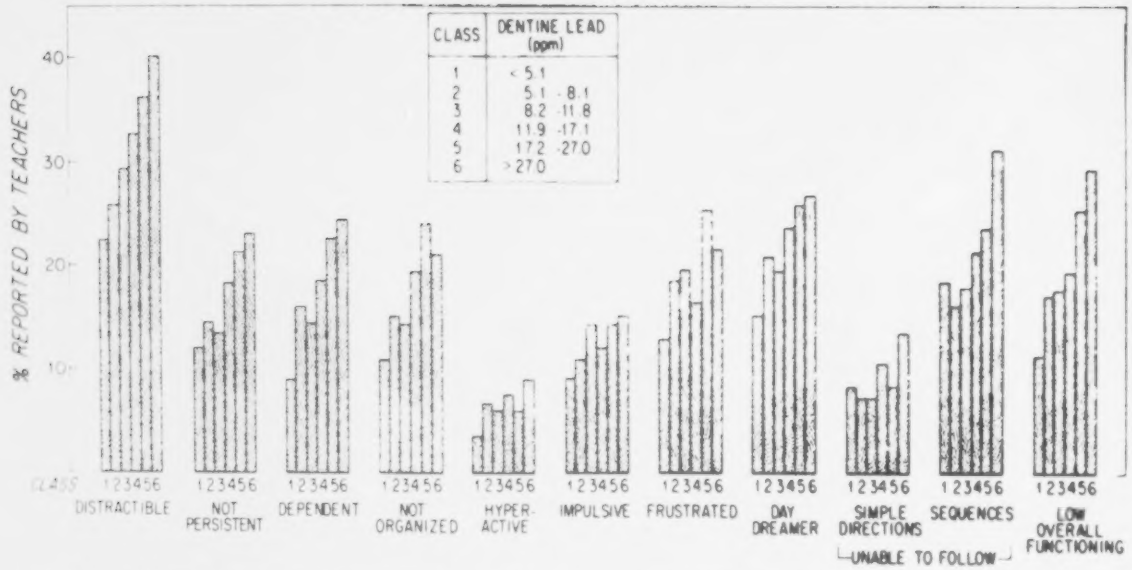

Figure 1. **Teachers' Ratings of Classroom Behavior in Relation to Dentine Lead Level.** The height of each bar represents the percentage of negative reports for each lead group.

Prevalence of lead exposure

The Agency for Toxic Substances and Disease Registry (ATSDR) of the Public Health Service compiled the most recent and authoritative epidemiologic data on lead exposure in 1987 (6). Having determined that neurotoxicity begins at blood levels as low as 10-15 ug/dl, ATSDR used data from the NHANES II study of child health, which measured blood lead levels from more than 5,000 American children between 6 months and 5 years of age. ATSDR has estimated that between three and four million children have blood leads greater than 15 ug/dl. This means that one American child in six has toxic amounts of lead in his or her blood, and that 400,000 newborns each year are delivered bearing toxic levels.

ATSDR has reported to Congress that 20 million homes in the U.S. have lead painted surfaces. There are two million homes which are deteriorated, and in which children live. These are the twentieth century equivalent of pesthouses or open sewers.

As with many other health threats, lead exposure is not equally shared. Fifty-five percent of black children in poverty have blood lead levels greater than 10 ug/dl! This datum is one of the greatest single threats to American society.

Primary prevention: A modest proposal

Enormous progress has been made in the scientific understanding of the neurobiology and epidemiology of lead exposure. This progress has been reflected in public policy, although not without intense fights. An air standard was written, and over the past decade 90 percent of the lead added to gasoline has been removed. Blood lead levels in newborns have dropped in parallel fashion. This is a public health triumph.

But what of the most important source of hazardous exposure to lead—old housing? Little or nothing has been accomplished here. The responsibility for inaction must be borne by slum landlords, by banks that will not give home improvement loans, and, most bitterly, by the federal agency established to help house the poor, the Department of Housing and Urban Development (HUD).

HUD has been a regressive force in lead abatement. The title of a General Accounting Office report issued more than a decade ago constitutes a succinct indictment: "HUD Not Fulfilling Responsibility to Eliminate Lead-Based Paint Hazard in Federal Housing" (20). If one is puzzled by the disparity between the scientific progress made over the past 15 years and the absence of progress in lead paint removal, one has to look first at HUD and its contractors.

If one were to map the prevalence of excess lead burden, and map the prevalence of housing shortages, and map the prevalence of unemployment, these maps would be isomorphic. What could be done to rationalize this disequilibrium of risk? Why not train unemployed persons from high lead areas in safe (and inexpensive) deleading and housing rehabilitation, pay them a decent wage, and allow them to purchase equity in deleaded houses? Costs of deleading a small house are estimated at $5,000. To delead 2 million homes would cost $10 billion over a decade. This figure would include employing 30,000 individuals at $20,000 per year, with a five percent annual increase, (a total of $7.8 billion) and spending $2.2 billion for training, materials, and insurance. Of the total sum, approximately $1.5 billion would be returned to the government in taxes. Money paid in salaries would be likely to circulate in the inner city neighborhoods where the workers were employed, thereby multiplying its impact. If this plan sounds Utopian, one needs to be reminded that the current request for appropriations for new prison construction is $11.6 billion.

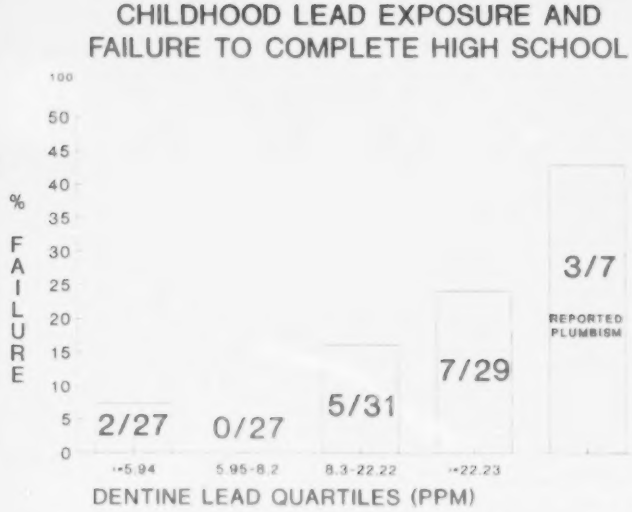

Figure 2. **The proportion of subjects who failed to graduate high school classified by past exposure to lead.** Asymptomatic subjects are classified by lead quartile. Seven of 10 subjects who were reported to have had clinical plumbism are also displayed. School records were not found for two subjects. One subject was not tested, but personally reported that she had graduated high school. Ten subjects are still attending high school and are not shown here. Three of these are in the group with reported plumbism and seven are asymptomatic subjects.

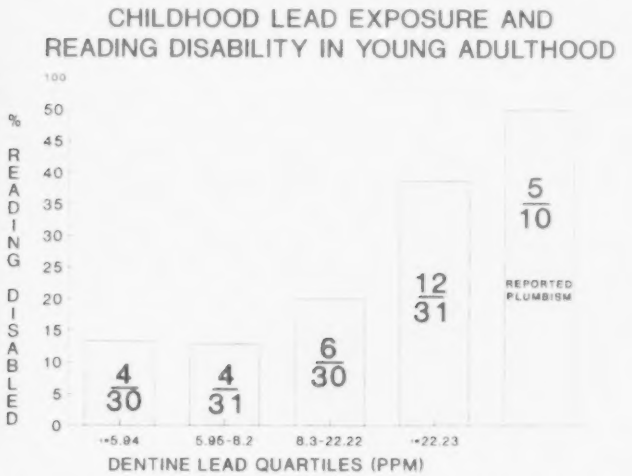

Figure 3. **The proportion of reading disabled children classified by past exposure to lead.** Asymptomatic subjects are classified by lead quartile, and ten children with a past history of clinical plumbism are displayed. Reading disability is defined as reading at two or more grades below expected.

In 1963, the Executive Secretary of the World Health Organization was approached with a plan to eradicate smallpox from the earth. He scoffed. Because he believed that the project was doomed, he requested that an American be asked to direct the effort (and ultimately take the blame for failure). Dr. Donald Henderson and the Centers for Disease Control accepted the challenge. Today, smallpox is a disease for the history books.

Lead poisoning, the silent, relentless destroyer of brain cells and of children's futures, can have the same fate as smallpox. All we need to do the job are vision and commitment.

The Alliance to End Childhood Lead Poisoning

The Alliance to End Childhood Lead Poisoning is a new national public interest organization, established by leaders in American pediatrics, public health, environmental protection, low-income housing, education, and children's welfare. The mission of the Alliance is to change public perceptions about childhood lead poisoning; expand the resources available at the federal, state and local levels for prevention of childhood lead poisoning; and develop and implement effective national prevention programs.

Herbert L. Needleman is Chairman of the Alliance to End Childhood Lead Poisoning. The National Center for Clinical Infant Programs is a member of the Alliance.

Three projects constitute the Alliance's current agenda:

- A national conference on October 7-8, 1991 (see *Conference Call*, p. 29) which will bring together individuals from the fields of medicine, public health, education, housing, lead abatement, and finance to develop a national agenda for action;
- Development of a handbook of strategies to help local programs respond to the increased number of children who will be identified as lead poisoned under anticipated new CDC guidelines; and
- Serving as a national resource for referrals and materials related to the prevention of lead poisoning.

For educational and technical materials, tips for parents and homeowners, and additional information about preventing childhood lead poisoning, please send a self-addressed stamped envelope to:

Alliance to End Childhood Lead Poisoning
600 Pennsylvania Avenue, S.E., Suite 100
Washington, D.C. 20010

Don Ryan, Executive Director of the Alliance, may be reached at tel: (202) 543-1147, FAX: (202) 543-4466.

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Biochemical actions of lead

It is not known which of lead's multiple biochemical actions is the basis of its toxicity. The known actions include:

- **binding to the sulfhydryl groups of proteins (Valle, B.L. & Ulmer, D.D. [1972] Biochemistry of lead. In: *Annual Rev. Biochem.* E.E. Snell (Ed.) 2/1:p.91.)**
- **catalytic cleavage of RNA during translation (Brown, R.S., Hingerty, B.C., Dewan, J.C. & Klug, A. [1983] Pb(II)-catalysed cleavage of the sugar-phosphate backbone of yeast tRNA Phe-implications for lead toxicity and self-splicing RNA. *Nature;* 309:543-546.)**
- **activation of phosphokinase C (Markovac J., & Goldstein, G.W. Picomolar concentrations of lead stimulate brain protein kinase C [1988] *Nature;* 334: 71-73.)**
- **inactivation of opioid receptors (Bailey, C. & Kitchen, I. Ontogenesis of proenkephalin products in rat striatum and the inhibitory effects of low-level lead exposure [1985] *Dev. Brain Res.;* 22: 75-79 and Kitchen, I., McDowell, J., Winder, C., & Wilson, J.M. [1984] Low-level lead exposure alters morphine antinociception in neonatal rats. *Toxicology Letters;* 22:119-123.)**
- **and others (Nation, J.R., Baker, D.M., Fantasia, M.A., Ruscher, A.E. & Clark, D.E. [1987] Ethanol consumption and free operant avoidance performance following exposure to dietary lead. *Neurotoxicology;* 8:561-568.)**

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Research in Process: A Comparison of Lead Screening Methods in Young Children

by

Catherine S. Rude, M.D. and David Schonfeld, M.D.
Yale University School of Medicine, Department of Pediatrics

Attention to the problem of lead poisoning in the United States is increasing. All proposals designed to eliminate the threat of lead poisoning in children include screening for lead poisoning as a major part of any effort.

The Centers for Disease Control (CDC) currently recommend screening all children considered to be at risk between 9 months and 6 years of age. CDC guidelines currently recommend medical treatment and public health measures when a child's venous lead level (level of lead in the blood) reaches 25 ug/dl (micrograms per deciliter). It is anticipated that the CDC will soon recommend that medical or public health measures be taken when the venous lead level is 10-15 ug/dl.

The "gold standard" test for lead poisoning is a venous lead level usually obtained from a vein in the arm. The CDC currently recommends (DHHS, 1985) as screening tests either free erythrocyte protoporphyrin (FEP or ZPP), easily tested with a fingerstick blood sample, or blood lead levels. As a screening test, a fingerstick blood sample is preferred over a sample from the vein, because it is easier to do, has greater acceptance by patients, and allows a larger number of children to be tested. The blood sample for FEP obtained by a fingerstick test is advantageous because it can be analyzed at the clinic site and the results reported immediately. Moreover, the FEP can be used to screen for iron deficiency anemia as well as lead poisoning

Unfortunately, the FEP test detects only about one-half of children with lead levels greater than 30 ug/dl and is even less effective at identifying children with lower lead levels (Mahaffey, KR and Annest, JL, 1986). Thus the usefulness of the FEP test as a screening tool is questionable under either current or anticipated CDC guidelines.

Some states have recently instituted lead screening programs using fingerstick lead tests. This involves collecting blood obtained from a fingerstick on a piece of filter paper or in a small capillary tube. These samples are then sent to a central laboratory for analysis. While this kind of screening can be done on a large scale, the blood samples are often contaminated with lead-containing dust from the child's finger, resulting in a large number of false positive tests. Notification of results often takes several weeks. Because of the finger lead contamination and the lag time for notification, many children may need to be called back to the office or clinic for confirmatory venous blood lead tests. Contacting the family and arranging for a follow-up test require time and effort, scarce resources in many clinic settings.

Many children are not currently screened for lead poisoning by any method of blood sampling because they are not considered to be at risk. We do not know how many cases of lead poisoning are missed through failure to screen. Asking parents about certain risk factors for lead poisoning may be a promising method of screening. Questions would examine such areas as: 1) a child's exposure to peeling paint; 2) the occurrence of lead poisoning in other family members; and 3) exposure to industrial or occupational sources of lead.

At the Yale University School of Medicine, we are comparing three methods of screening for lead poisoning: 1) fingerstick FEP; 2) fingerstick lead (filter paper or capillary); and 3) a lead screening questionnaire. We plan to compare the efficacy of these methods at detecting both $\geq$25 ug/dl and $\geq$10-15 ug/dl lead levels.

We plan to obtain approximately 500 screening samples over a three-month period at the Yale-New Haven Hospital Primary Care Center, a pediatric clinic that serves inner city children. These samples will be analyzed for FEP on site, sent to the hospital laboratory for capillary lead assessment, and sent to the Connecticut Health Department Laboratory for filter paper lead measurement. A research assistant will complete the lead screening questionnaire for each child screened. Venous lead levels will be obtained at the time of screening in children with an elevated FEP, or following the report of an elevated ($\geq$25 ug/dl) filter paper or capillary lead level. Parents whose children show filter paper or capillary lead levels between 15 and 24 ug/dl will be approached for consent for a venous lead level. We will record the amount of time clinic personnel or the research assistant spends in bringing children with positive screening results back to the clinic for venous lead determinations.

Our data will allow us to determine the false positive rates for FEP and filter paper/capillary lead, and to begin to examine the effectiveness of a lead screening questionnaire. We will also be able to compare the various screening methods in terms of the time spent in confirming positive test results. We hope to be able to compare the utility of these various methods as screening tests for lead poisoning both at currently defined levels and at anticipated levels. As proposed plans to eliminate lead poisoning in the United States move forward, we hope that our findings will increase the effectiveness of screening programs for children.

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Childhood Burns: The Preventable Epidemic

by

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Every year in the United States, hundreds of infants and toddlers die of burns. Thousands of other very young children survive burn injuries, but at a cost of untold suffering. In the United States in 1985, approximately 12,500 children 0-4 were hospitalized for burn injuries, and 132,000 more suffered burns severe enough to require medical attention (McLoughlin & McGuire, 1990).

A severe, nonfatal burn is the most devastating injury the human body can survive. When a young child is burned severely, physical and emotional suffering can be lifelong. The initial pain of the burn itself is followed by excruciating treatment procedures. Children are frequently separated from their parents and siblings during extended and repeated hospitalizations. Disfigurement and/or physical disabilities may be permanent, and children who were severe burn victims are more likely than their counterparts to have emotional problems (Stoddard, Norman, Murphy, & Beardslee, 1989).

Most burns to young children can be prevented. As we understand the reasons for the high incidence of burns among young children in this country, we can develop and implement intervention strategies to reduce the number and severity of burn injuries to children. In recent decades we have seen major advances in burn prevention. Further progress will not be easy—there is no single magic bullet—but there is no doubt that this epidemic is one that can be halted.

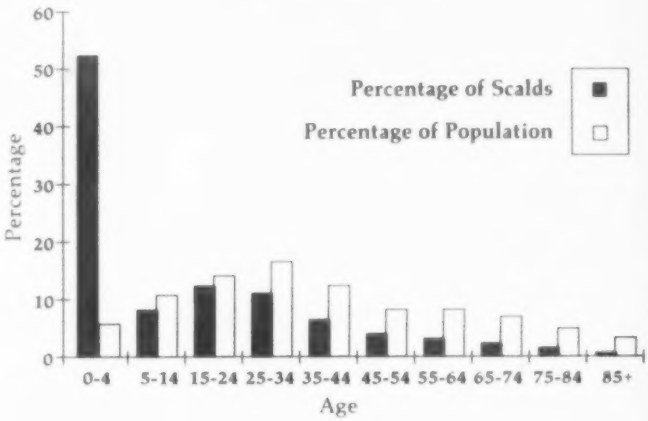
The epidemiology of burns in children

To understand the problem of childhood burns, it is useful to use a general "epidemiologic model," which focuses attention on three factors: *the host*, in this case the child; *the agent*, the direct cause of the injury—e.g., a match; and *the environment*, both physical and socioeconomic. To prevent burns among young children, we need to understand each of these factors, the interaction among them, and the opportunities available for change.

The child: Because they do not recognize danger or know how to protect themselves, babies and toddlers are prone to injuries of all sorts. Play and exploration are developmentally appropriate and important for young children, as are their drives toward imitation and autonomy. But the inability of young children to comprehend dangerous consequences of their actions makes them vulnerable to injury. In addition, young children are less able than adults to escape from a dangerous situation, such as a house fire or a bathtub full of scalding water. Once exposed to an injurious agent, children's smaller body size and more sensitive skin make them more vulnerable than adults to death, disability and disfigurement. Consequently, young children as a group experience particularly high rates of death and disability from all types of injury, including burns.

The agent: Children are burned most frequently by scalds, and most severely by house fires. Seventy per cent of children 0 to 4 years old who were hospitalized for burns in 1985 had suffered scalds. Eighty-five percent of burn deaths in this age group were due to house fires (McLoughlin & McGuire, 1990).

Scald Injuries by Age and Percentage of Population, Massachusetts 1988



1988 figures from the Massachusetts Burn Injury Reporting System (Figure 1) demonstrate the high incidence of scalds in young children, especially compared to other age groups. Scald burns occur primarily in the kitchen and in the bathroom, with bathroom scalds tending to be much more severe (McLoughlin & Crawford, 1985). In the kitchen, the typical incident involves a young child pulling a cup or pot of hot water onto himself, or a parent spilling hot liquid onto a child. Bathroom scalds typically occur when children are placed in a bathtub in which the water is too hot or when the hot water is turned on a child full force. Children who live in apartment complexes are at particularly high risk of scald burns in the bathtub because the temperature of water heaters serving large buildings is frequently set extremely high in order to provide hot water to tenants on the top floors. Especially severe scald burns have been seen in children who live on the bottom floor of high-rise buildings (McLoughlin & Crawford, 1985).

Children and the elderly are at greater risk of death from house fires than any other age groups (Baker, O'Neill & Karpf, 1984). Thirty to 45 percent of these deaths are the result of cigarette use by adults. Heating

BARBARA YOUNG

equipment and electrical malfunction are the second and third leading ignition sources. Only about two percent of residential fire deaths are attributed to children playing with matches, cigarette lighters, or other ignition sources (McLoughlin & Crawford, 1985).

Other sources of burn injuries are clothing ignition, electrical burns, and contact burns. The use of flame-retardant fabrics has markedly reduced clothing ignitions in young children, as will be discussed below. Young children suffer electrical burns primarily when they suck or mouth plugged in extension cords (McLoughlin & Crawford, 1985). While these burns are not as severe as scalds and ignition burns, they do lead to some 4,000 emergency room visits per year, most of which involve young children. Burns from contact with floor furnace gratings and wood burning stoves are not high incidence occurrences.

The environment: The safety of a child's environment is a function of the adequacy of supervision (including the caregiver's knowledge of potential hazards) and the degree to which hazards have been eliminated from the environment. Injury prevention specialists have found that "passive" interventions, which make the environment safer for people, are more effective than "active" strategies, which require people to do something to protect themselves. Thus making cigarettes safer is likely to be more effective in preventing house fires than educational programs urging people to avoid smoking in bed.

Both passive and active strategies are needed. New products cannot be expected to prevent all burn injuries, and even when safer products are developed they will not immediately replace all older, less safe models. To prevent burns in children, parents need to be aware of potential dangers in the environment and ways that they can make the environment safer for the child.

Simple solutions are not always available. For example, hot liquids in the kitchen cause a major proportion of burns to young children. A child may pull a pot of hot water off the stove or trip a parent who is carrying a pot of boiling water. How can we reduce the occurrence of these incidents? It is one thing to emphasize to parents the importance of always turning a pot handle away from the front of the stove, so that a toddler cannot reach it. It is something else to suggest that toddlers be kept out of the kitchen during meal preparation to lessen the chance of scald burns. This strategy might result in a toddler's being unsupervised elsewhere in the house, and might deprive a parent and child of important, pleasurable time together.

Education for parents and other caregivers should stress burn treatment. Burn injuries can be significantly less severe if the burn is treated immediately with cold water. Butter or salves should not be applied as treatment.

Socioeconomic status is an aspect of a child's environment that is highly relevant to burn injuries.

Not surprisingly, poor children are at higher risk of burn injury (MacKay, Halpern, McLoughlin, Locke & Crawford, 1979); one study found an extremely strong association between poverty status and the incidence of hospitalization for burns (Locke, Rossignol, Boyle, & Burke, 1986). No research completely documents the links between poverty and burns, but it is easy to speculate: outmoded heating and bad wiring, and the use of space heaters in the homes of low-income families may lead to more house fires; smoke detectors may be viewed as a luxury; dangerously hot tap water may be more common in multi-unit low-income housing; and economic constraints may lead to inadequate adult supervision by parents or other caregivers.

Preventing burns to children

Contemporary attitudes toward injury resemble nineteenth century ideas about infectious disease. Before the natural causes of infectious disease were understood, such illness was viewed either fatalistically or as the consequence of individual moral failings. Today we tend to view "accidents" in the same way. As long as we continue to perceive injuries as either inevitable or due to individual misbehavior, we will not devote sufficient energy to devising and implementing injury prevention strategies that work. A comprehensive approach focused on product, environmental, behavioral, and socioeconomic/cultural changes can be effective in preventing injury, as it has been in preventing disease.

Preventing burns through product changes: Sometimes relatively simple changes in products can have significant effects in preventing burn injuries and deaths. Flame-retardant fabrics, hot water heaters and anti-scald devices, and fire-safe cigarettes are good examples of the issues involved.

- **Flame-retardant fabrics:** In the early 1970's, the Consumer Product Safety Commission promulgated standards to reduce the flammability of children's sleepwear. Prior to the promulgation of these standards, a child playing with a match or getting too close to a stove could be severely burned when his pajamas went up in flames. Advocates argued that children's sleepwear could be made from materials that retarded fire. These advocates eventually prevailed, and sleepwear for children's sizes 0 to 14 must now meet minimum standards for flammability.

A study at one pediatric burn unit in Boston found a striking difference in the types of burn injuries suffered by children before and after these standards were implemented (McLoughlin, 1977). Prior to the introduction of flame-retardant sleepwear, a high percentage of admissions to the burn center were due to ignition of sleepwear. By 1976, when only flame-retardant sleepwear was being manufactured, only one child was admitted to the unit with such burns, and in that case the flames had spread slowly and burns were confined. Moreover, the burn center treated two infants that same year who had been burned in house fires, but who had been spared burns on all parts of their bodies that had been covered by flame-resistant sleepwear.

Today, very few children are seen in hospitals for burns related to sleepwear ignition. The evidence suggests that legally mandated flame-retardant sleepwear is an intervention that protects many children each year from painful, severe burns.

- **Hot water heaters and anti-scald devices:** About five thousand young children each year are burned severely from hot tap water (Bacon, 1990). Tap water burns are generally more extensive and more severe than other scald burns; they can cause permanent disfigurement or death (Katcher, 1981). Very young children, the elderly, and individuals with disabilities are the groups most likely to suffer severe tap water burns. In one Wisconsin county over a ten-year period, 52 percent of all tap water scalds severe enough to require hospitalization were suffered by children under five (Katcher, 1981).

Young children are burned by tap water in several typical scenarios. Left alone for a few seconds in a bath, they may turn on the hot water themselves. An older sibling may "help" wash the baby by turning on the water. A third cause of scalds is child abuse: most commonly, immersion burns are caused by forcibly placing a child in a tub of hot water (Stuart, Kenney & Morgan, 1987) or running very hot water over a child's buttocks, perhaps as a punishment in toilet training. In fact, one study found that some 28 percent of the tap water scalds severe enough to require hospitalization were due to abuse (Feldman, 1978).

Historically, hot water heaters have been preset at temperatures so high that a 2- to 5-second exposure could lead to serious burns in an adult. If the temperature on hot water heaters is set above 130 degrees, babies and young children are at risk of being burned. Babies and toddlers are usually not able to escape from hot water. And because their skin burns faster, children sustain serious burns at lower temperatures and with shorter exposure than adults.

Advocates have worked for years on behalf of laws mandating lower preset temperatures on new hot water heaters. Florida enacted such a law in 1980, Washington state in 1982, and Wisconsin in 1988. Following the enactment of Washington's law, the number of children brought to Seattle clinics with tap water burns fell by half (Meier, 1990). Faced with the prospect of further state action, manufacturers have now begun voluntarily to pre-set heater temperatures at safe levels. We can expect to see the incidence of tap water scald burns decrease dramatically over the next ten to fifteen years as older water heaters are replaced with new ones, preset at lower temperatures.

Programs have also been developed to encourage people to turn down the dangerously high temperature levels of hot water heaters purchased before new standards were in place. In Wisconsin, a media campaign informed people of the danger of high heater settings and made available free thermometers for testing the

What Can You Do to Prevent Pediatric Burns?

At home—

- **Install and maintain smoke detectors on every floor. Test the detectors frequently, and replace the batteries at least once per year.**
- **Turn down the hot water heater thermostat to 120 degrees Fahrenheit or install anti-scald devices in faucets and shower heads.**
- **In the bathroom, test the water by swishing your hand with fingers spread apart before placing your child in the bathtub. Never leave a child unsupervised. If the telephone or doorbell rings, take the child with you.**
- **In the kitchen, use the rear burners and turn pot handles toward the rear of the stove. Do not hold a child while drinking a hot beverage. While cooking, keep young children in high chairs or playpens.**

In your community, support—

- **Smoke detectors in all homes.**
- **Fire-safe cigarettes.**
- **Sprinklers in new home construction.**
- **Enforcement of building codes.**
- **Adequate and affordable child care.**
- **Comprehensive burn prevention programs which make smoke detectors and anti-scald devices available to those who cannot afford to purchase them.**

temperature of water. This campaign resulted in 20,000 water heater set-backs; 52 percent of the people who received the free thermometers and who had dangerously hot tap water lowered the temperature to safe levels. While this is a significant achievement, it is disheartening to note that so many people did nothing, even when confronted with this danger in their homes. While educational campaigns can be effective, the limitations of "active" strategies of prevention are vividly illustrated here.

Another partial solution to the tap water scald problem is the insertion of an inexpensive device in faucet heads which expands when water reaches a certain temperature and blocks the flow of water. This strategy is especially useful in apartment buildings where water must be heated to a very high temperature in order to provide hot water to all apartments, putting families in apartments near the heater at risk for severe burns. Last year, the advocacy group SAFE KIDS installed anti-scald devices in multi-unit, low income dwellings in several cities.

In sum, tap water burns are preventable through technological changes as simple as lowering the temperature setting of water heaters and installing anti-scald devices in faucets. These changes can even reduce abuse-related burns to children, by making it significantly more difficult for a potential abuser to burn a child. It is likely that many parents have burned a child in anger who truly did not intend to harm the child, or at least not to the extent possible with very hot tap water. Unfortunately, children will continue to be severely burned by tap water, unintentionally and intentionally, until all old water heaters are replaced by safer models and until anti-scald devices are installed where temperature settings on heaters remain dangerously high.

• **Fire-safe cigarettes:** Children and the elderly are at greater risk of death from house fires than any other age groups (Baker, O'Neill & Karpf, 1984). Since some 35 percent of all these fires involve cigarettes (Consumer Product Safety Commission, 1987) children's deaths would be reduced dramatically by "fire-safe" cigarettes. These are cigarettes designed in such a way that when left unattended on mattresses and upholstered furniture, they will not cause ignition.

The National Bureau of Standards began research on fire-safe cigarettes in 1929 and developed prototypes in 1932. However, no manufacturer introduced such cigarettes voluntarily, and no laws compelled them to do so. Andrew McGuire, Executive Director of the Trauma Foundation at San Francisco General Hospital became so outraged by needless deaths and severe burn injuries that in 1978 he began a national grassroots campaign for legislation to mandate fire-safe cigarettes. By developing a coalition of fire fighters, physicians, anti-smoking groups, and others, he succeeded in having such legislation introduced into 13 state legislatures. Although none of these bills has passed, the advocacy effort raised liability concerns for cigarette manufacturers and led to their support of national legislation, the Fire Safe Cigarette Act of 1990, to develop standards for fire-safe cigarettes by 1993. Once these standards are developed, it is anticipated that a new law mandating their application will be enacted quickly and without difficulty. Thus, by the mid-1990s we can expect, finally, to have fire-safe cigarettes.

It is estimated that this simple technological innovation will save up to 1500 lives (including the lives of 500 children) each year, and prevent 6,000 injuries annually. The example of fire-safe cigarettes illustrates the impact that a committed person, working with a coalition, can have, and it demonstrates once again that fires and burns can be prevented. However, it is tragic that technology available since 1932 has yet to be adopted, and that the lives of thousands of children have been needlessly lost or filled with suffering.

Preventing burns through environmental modifications—the smoke detector: Another strategy for preventing injury is to make the environment itself safer. Smoke detectors are an example of an environmental modification.

Installation and maintenance of smoke detectors constitute a proven strategy to prevent death and injury from house fires (McLoughlin et al., 1985). The United States Fire Administration reports that 90 percent of child fire deaths occur in homes that do not have a

working smoke detector (USFA, n.d.). While smoke detectors do not prevent fires, they do provide an opportunity for escape. The risk of being killed by a fire in a home without a smoke detector is two and one-half times the risk of being killed in a home with one (Gorman et al., 1985).

Montgomery County, Maryland, was the first major jurisdiction to pass a law mandating smoke detectors in all homes. This action led to a measurable decline in deaths due to fires (McLoughlin et al., 1985). By 1988, approximately 82 percent of U.S. homes and apartments had smoke detectors, but because about one-third of these lacked working batteries, many homes remain unprotected. Effective strategies to use smoke detectors to improve safety include:

- requiring new dwellings to have electrically powered smoke detectors rather than battery-operated models;
- outreach to high-risk areas for inspection of detectors and fire hazards; and
- education campaigns.

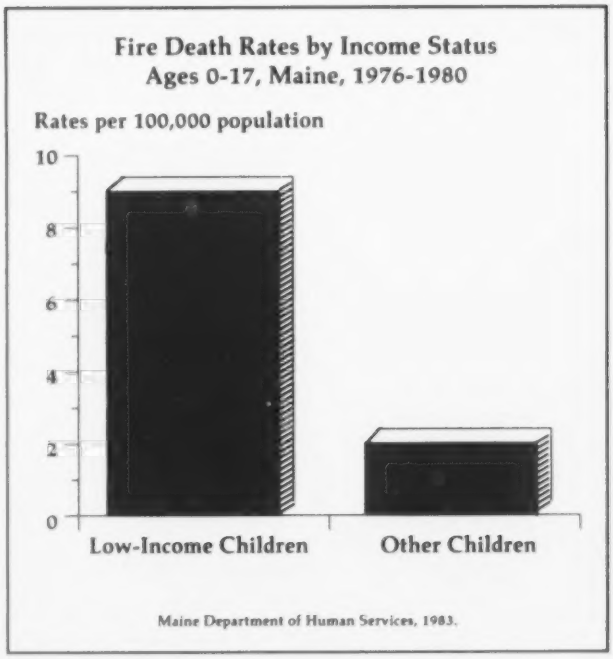
This example illustrates the need for continual monitoring to maintain the effectiveness of burn prevention efforts.

Preventing burns through socio-environmental strategies: As noted earlier in this article, children in poverty are at higher than average risk of burn injury, just as they are at higher risk for health problems in general. Figure 2 illustrates the significant difference poverty can make in child fire deaths. The supervision differential may be extremely critical in fire deaths among low income children. Economic pressures send parents and adult relatives into the out-of-home work force, and adequate child care may be unavailable or unaffordable. Children who should be in supervised care themselves may be responsible for babies and toddlers. Newspaper stories describing house fires set by unsupervised children are all too frequent. Nationally, eight percent of all fire deaths are the result of children playing (U.S. Fire Administration, 1989).

Inadequate supervision of children may also be part of a family picture that includes mental illness or substance abuse. Too often, treatment for adults fails to take into account their parental roles or the needs of their children. Services that recognize and support the parenting role of troubled adults and that include enhanced supervision for children could do much to prevent burns and other injuries while improving the lives of children in other ways as well.

A comprehensive burn prevention program

New York State is presently conducting a three-year comprehensive burn injury prevention program, funded by the federal Maternal and Child Health Bureau and focusing on rural low-income families. Burn prevention activities are coordinated with local Weatherization Assistance Programs. While Weatherization Assistance Programs are designed primarily to help families save money through energy conservation, they may also be a vehicle for making homes safer—for example, by lowering tap water temperatures.



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The joint weatherization assistance/burn prevention program appealed to state policymakers for several reasons:

1) Weatherization serves low-income families, who are at highest risk for burns;

2) Weatherization Assistance Program staff have easy access to many households and are themselves representative of the low-income target population; and

3) the energy audit performed by the program is easily extended to include a "burn safety audit," which includes assessing and, if necessary, reducing tap water temperatures, checking electrical systems for dangers, checking for the presence of functional smoke detectors, and testing and repairing heating systems.

The Weatherization staff is trained to provide consultation to families on burn prevention measures. If major deficiencies are found in any of the checked systems, they are corrected by professional building contractors without charge to low-income families. Finally, the Weatherization staff provides follow-up inspections and evaluations of fire-related home safety hazards. As of October, 1990, the New York State program had conducted comprehensive room-by-room surveys in several thousand homes, installed more than 9,000 smoke detectors, lowered temperatures on 1,000 water heaters, and installed 75 anti-scald devices. In addition, staff had distributed more than 4,000 burn injury prevention kits and counseled several thousand

families on fire prevention and basic fire safety messages (e.g.,"If your clothes catch fire, stop, drop and roll.")

This program demonstrates an approach to injury prevention incorporating both "active" and "passive" interventions targeted toward a particularly high-risk population.

Conclusion

We have seen successful efforts to prevent burns in young children. Flame-retardant sleepwear and smoke detectors save lives and prevent injury. Comprehensive approaches, like the New York State Weatherization Assistance Program, show promise as imaginative combinations of active and passive strategies. By carefully identifying the circumstances and mechanisms that lead to children's burns, we can design effective programs to reduce pediatric burn injuries significantly. This is an epidemic that we can prevent, if we have the will to do so.

For additional information about pediatric burn injuries, please contact the following resources:

Children's Safety Network
38th and R Streets, NW
Washington, DC 20057
(202) 625-8400

National SAFE KIDS Campaign
111 Michigan Avenue, NW
Washington, DC 20016
(202) 939-4993

New York State Department of Health
Injury Control Program
Corning Tower, Room 621
Empire State Plaza
Albany, NY 12237
(518) 473-1143

The Trauma Foundation
San Francisco General Hospital
Building One, Room 311
San Francisco, CA 94110
(415) 821-8209

U.S. Fire Administration
17825 South Seton Avenue
Emmitsburg, MD 21727
(301) 447-1181

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Infants, Toddlers, and Indoor Air Pollution

by

Nancy Thorndike Greenspan

Infants and toddlers are at greater risk from indoor air pollution than older children or adults. Both the developing physiology of very young children and their different physical environments contribute to this vulnerability.

The World Health Organization (1986) lists seven reasons why children may be more vulnerable to toxins than adults:

- larger body surface area in relation to weight;
- higher metabolic rate and oxygen consumption per unit of body weight;
- different body composition;
- greater energy and fluid requirements per unit of body weight;
- special dietary needs;
- rapid growth, during which chemicals may affect growth or become incorporated into tissues; and
- functionally immature organs and body systems.

Of course, children who are in poor health or have certain disabilities may be affected more seriously by toxins than their healthy counterparts.

The results from animal studies (Calabrese, E.J., 1986; Klaasen, C.D., 1986; World Health Organization, 1986; Marquis, J.K. and Siek, G.C., 1988) suggest that the young retain many chemicals or do not detoxify them efficiently. Their gastrointestinal membranes are more permeable than those of adults; their kidneys and enzymes are less capable of detoxifying chemicals; and their blood-brain barriers are less fully developed. These differences would result in children having higher blood levels of certain toxins than adults with the same exposure and greater susceptibility to carcinogens and neurotoxins.

The development of the young child's nervous system is particularly vulnerable. According to a 1990 statement of the Office of Technology Assessment

> When the developing nervous system is exposed to neurotoxic substances, the process by which cells multiply and connect may be disrupted, leading, for example, to the severe retardation seen in children exposed to high levels of mercury, or perhaps to more subtle deficiencies related to learning and memory, or to neurological disorders. (pp. 4-5).

Young children also inhabit physical environments different from those of older children and adults. Because they cannot go outside independently, they are often indoors more than older children. Infants and toddlers spend a lot of time in cribs and cots, and on, or close to, the floor. Consequently products used on bedding (fabric dyes, detergents, bleach) and the floor (carpeting, cleaning compounds) are more significant for the young child. So are pollutants that concentrate at ground level (radon, mercury from latex paint, heavier particles from environmental tobacco smoke) and dust that is contaminated by these pollutants.

Unfortunately, parents, child care providers, and administrators with the authority to regulate children's physical environments have little awareness of children's health risks from indoor air pollution. Little documentation exists, and the effects of chronic exposure to low levels of toxins—the situation children most often face—have not been studied.

This article will review our general understanding of the health effects of indoor air pollution; consider some specific pollutants; discuss the issue of chemical sensitivity; and suggest sources of additional information and areas for further research.

Sources of indoor air pollution

Thousands of natural and synthetic air pollutants can be present indoors. Some, such as radon (the gas produced by decomposing uranium) or carbon monoxide, seep into buildings from the ground or the outdoor air. Other pollutants, such as formaldehyde and methylene chloride, "outgas" (are emitted) from building materials themselves or cleaning products. We consciously introduce some indoor air pollutants by smoking or spraying pesticides. Biological contaminants such as molds or bacteria grow in, and are subsequently emitted by, heating and air conditioning systems.

Almost any inhabited building—home, child care center, school, library, store, restaurant, or office—contains some toxins. Although some occur naturally, many have been created by human activity.

The living room fireplace, for example, needs consideration along with the industrial smokestack. Wood smoke, composed of those gases and particles arising from fireplaces and wood stoves, contains some of the dioxins, one of which (2,3,7,8-TCDD) is best known as a major toxin in Agent Orange. Dioxins are not quickly eliminated from the body, but accumulate in fat tissue.

Some researchers wondered whether dioxins were a natural component of wood or an artificial contaminant (Okie, 1989). To answer the question, they examined the muscle tissue of nine 2,800 year-old mummies from the northern Chilean desert. If dioxins were naturally present in wood, the researchers hypothesized, people cooking over wood fires thousands of years ago would have been exposed to dioxins and would have accumulated them in their body fat, as we do.

Since the researchers found no dioxins in the mummies' muscle tissue, they concluded that wood combustion before the industrial age did not release dioxins. They suggested that as trees work to filter our air, they absorb modern-day pollutants; these turn to dioxins as the wood is burned.

BARBARA YOUNG

Ours is hardly the first generation to experience the effects of indoor air pollution. The Mad Hatter in Alice in Wonderland reminds us that in the nineteenth century, makers of beaver hats, who used mercury in their trade, suffered central nervous system damage from the chemical (Neal, P.A. & R.R. Jones, 1938). It is disturbing to discover that mercury was an ingredient in 25 to 30 percent of all indoor latex paint until 1990. A government investigation of children's motor problems and skin irritations traced problems to the mercury in paint, causing the Environmental Protection Agency (EPA) to forbid its use. Mercury remains an ingredient in other building products such as spackling compounds, joint compounds, adhesives, and acoustical plasters (Indoor Air News, 1990).

Pollutants are not only introduced into indoor environments; increasingly they are trapped there—along with us. We spend less time than our ancestors did outdoors, and we have impeded outdoor air flow in our homes and workplaces to ensure greater "energy efficiency." In the last 10 to 15 years, particularly since the first oil crisis of the 1970s, three factors have interacted to create "sick building syndrome," an identifiable (but not always remediable) health problem. These factors are: 1) a decrease in air flow from the outside; 2) an increase in sources of pollution indoors; and 3) a failure of indoor air filtering systems to compensate for the first two factors.

"Sick building syndrome" encompasses many different symptoms, including fatigue, headaches and respiratory problems. The syndrome usually becomes identified in the work place when a disproportionate number of employees become bothered by these symptoms. (Ironically, a renovated building used by the federal Environmental Protection Agency has been so plagued by sick building syndrome that a number of the agency's employees now work at home to avoid feeling ill at work.) Indoor air pollution can produce similar health effects in houses and schools. However, the smaller number of people exposed in such instances and the difficulty of identifying the syndrome mean that indoor air pollution is often overlooked as a cause of health problems.

Specific pollutants

The Environmental Protection Agency's guide to indoor air quality (1988) lists 11 major categories of indoor air pollutants in the home: radon, environmental tobacco smoke, biologicals, carbon monoxide, nitrogen oxide, respirable particles, organic gases, formaldehyde, pesticides, asbestos, and lead (see Table 1).

Two other categories are potential additions to the list. One is the very controversial area of electromagnetic fields. Researchers are just beginning to examine cancer risks from high voltage wires, as well as from computers, electric blankets and microwave ovens (EPA, 1990). A second category is ozone. Photocopiers, fax machines, and laser printers are a source of ozone; measurements in offices show ozone levels up to ten times higher than the level considered safe (Allen, F.E., 1991). Electronic air filters are another source of ozone in homes and public buildings.

Any one pollutant, such as gasoline or smoke, is likely to contain hundreds of different chemicals that may be toxic. The research challenge is to discover at what level of exposure a pollutant may cause what kind and degree of harm to what kinds of children and/or adults.

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TABLE I - Summary of Major Household Pollutants

Pollutants	*Sources*	*Health Effects*	*Levels in Homes*
CARBON MONOXIDE	Unvented kerosene and gas heaters; leaking chimneys and furnaces; downdrafting from wood stoves and fireplaces; gas stoves. Automobile exhaust from attached garage. Environmental tobacco.	At low concentrations, fatigue in healthy people and chest pain in people with heart disease. At higher concentrations, impaired vision and coordination; headaches; dizziness; confusion; nausea. Can cause flu-like symptoms that clear up after leaving home. Fatal at very high concentrations.	Average levels in homes without gas stoves vary from 0.5 to 5 parts per million (ppm). Levels near properly adjusted gas stoves are often 5 to 15 ppm and near poorly adjusted stoves may be 30 ppm or higher.
ORGANIC GASES	Household products including: paints, paint strippers, and other solvents; wood preservatives; aerosol sprays; cleansers and disinfectants; moth repellents and air fresheners; stored fuels and automotive products; hobby supplies; dry cleaned clothing.	Eye, nose, and throat irritation; headaches, loss of coordination, nausea; damage to liver, kidney, and central nervous system. Some organics can cause cancer in animals; some are suspected or known to cause cancer in humans.	Levels of several organics average 2 to 5 times higher indoors than outdoors. During and for several hours immediately after certain activities, such as paint stripping, levels may be 1000 times background outdoor levels.
FORMALDEHYDE	Pressed wood products (hardwood plywood wall paneling, particleboard, fiberboard) and furniture made with these pressed wood products. Urea-formaldehyde foam insulation (UFFI). Combustion sources and environmental tobacco smoke. Durable press drapes, other textiles, and glues.	Eye, nose, and throat irritation; wheezing and coughing; fatigue; skin rash; severe allergic reactions. May cause cancer. May also cause other effects listed under "organic gases."	Average concentrations in older homes without UFFI are generally well below 0.1 (ppm). In homes with significant amounts of new pressed wood products, levels can be greater than 0.3 ppm.
PESTICIDES	Products used to kill household pests (insecticides and termiticides). Also, products used on lawns and gardens that drift or are tracked inside the house.	Irritation to eye, nose, and throat; damage to central nervous system and kidney; cancer.	Preliminary research shows widespread presence of pesticide residues in homes.
NITROGEN DIOXIDE	Kerosene heaters, unvented gas stoves and heaters. Environmental tobacco smoke.	Eye, nose, and throat irritation; may cause impaired lung function and increased respiratory infections in young children.	Average level in homes without combustion appliances is about half that of outdoors. In homes with gas stoves, kerosene heaters, or unvented gas space heaters indoor levels often exceed outdoor.
ENVIRONMENTAL TOBACCO SMOKE	Cigarette, pipe, and cigar smoking.	Eye, nose, and throat irritation; headaches; bronchitis; pneumonia. Increased risk of respiratory and ear infections in children. Can cause lung cancer and may contribute to heart disease.	Particle levels in homes without smokers or other strong particle sources are the same as, or lower than, those outdoors. Homes with one or more smokers may have particle levels several times higher than outdoor levels.
RESPIRABLE PARTICLES	Fireplaces, wood stoves, and kerosene heaters. Environmental tobacco smoke.	Eye, nose, and throat irritation; respiratory infections and bronchitis; lung cancer (Effects attributable to environmental tobacco smoke are listed elsewhere.)	Particle levels in homes without smoking or other strong particle sources are the same as, or lower than, outdoor levels.
LEAD	Automobile exhaust. Sanding or open-flame burning of lead-based paint. Activities involving lead solder.	Impaired mental and physical development in both fetuses and young children. Decreased coordination and mental abilities; damage to kidneys and nervous system, and red blood cells. May increase high blood pressure.	Lead dust levels 10 to 100 times greater in homes where sanding or open-flame burning of lead-based paint has occurred.
RADON	Earth and rock beneath home; well water; building materials.	No immediate symptoms. Estimated to cause about 10% of lung cancer deaths. Smokers are at higher risk of developing radon-induced lung cancer.	Estimated national average is 1½ picocuries per liter. Levels in homes have been found as high as 200 picocuries per liter. The higher the average concentration, the faster the corrective action should be taken. EPA believes that levels in most homes can be reduced to 4 picocuries per liter of air and sometimes less.
ASBESTOS	Deteriorating or damaged insulation, fireproofing or acoustical materials.	No immediate symptoms. Chest and abdominal cancers and lung diseases. Smokers are at higher risk of developing asbestos-induced lung cancer.	Elevated levels can occur in homes where asbestos-containing materials are damaged or disturbed.
BIOLOGICALS	Wet or moist walls, ceilings, carpets, and furniture; poorly-maintained humidifiers, dehumidifiers, and air conditioners; bedding; household pets.	Eye, nose, and throat irritation; shortness of breath; dizziness; lethargy; fever; digestive problems. Asthma; humidifier fever; influenza and other infectious diseases.	Indoor levels of pollen and fungi are lower than outdoor levels (except where indoor sources of fungi are present). Indoor levels of mites are higher than outdoor levels.

(Article continued after insert)

Although conventional wisdom has largely dismissed adverse health effects from low level exposures to many toxins, this complacency may prove to be dangerous. As Dr. Herbert Needleman (1990, and this issue) has found, levels of lead exposure previously considered "safe" have left many children with delayed mental development. Similarly, a recent study of workers at Oak Ridge National Laboratory (Wing, S., Shy, C.M., Wood, J.L., Wolf, S., Cragle, D.L. & Frome, E.L. 1991) found increased risk of leukemia from radiation levels well within U.S. safety guidelines.

The examples of three specific pollutants—environmental tobacco smoke, pesticides, and volatile organic compounds—illustrate some of the research, treatment, and policy challenges involved in indoor air pollution.

Environmental tobacco smoke

Environmental tobacco smoke (ETS), or "secondhand" smoke, is distinct from the "mainstream" smoke inhaled by the smoker. The combustion process that produces ETS is less complete than that which produces mainstream smoke, resulting in a 6 to 100 times greater concentration of carcinogens (Bearer, C.F., 1990).

ETS is not one chemical. Rather, it is made up of more than 4,700 chemical compounds, in both gas and particulate form. Many of these compounds are mutagenic, carcinogenic, or neurotoxic. The better known toxic components of ETS are carbon monoxide, nicotine, carcinogenic tars, sulfur dioxide, ammonia, nitrogen oxides, vinyl chloride, hydrogen cyanide, formaldehyde, benzene, and arsenic (EPA, 1989). (Interestingly, the presence of arsenic is due to its use as a pesticide on tobacco crops up to the mid-1940's. Its residues now persist in the soil, are absorbed by tobacco plants, and are emitted when the tobacco is burned [National Research Council, 1986]).

Anywhere from 8.7 to 12.4 million children are exposed to tobacco smoke at home (Office of Smoking and Health, 1986). Compared to children of non-smoking parents, young children of smoking parents have the following increased risks, according to the Office of Smoking and Health and the National Research Council (1986):

- 20% to 70% more hospitalizations for pneumonia and bronchitis;
- 20% to 80% more respiratory problems (wheezing, coughing, sputum production);
- more chronic ear infections, recurrent tonsilopharyngitis, and need for tonsillectomy and adenoidectomy;
- reduced growth and physical development; and
- increased risk of asthma;

Other researchers have found:

- increased risk for Sudden Infant Death Syndrome (Lewak, N., Van den Berg, B.J., Beckwith, B., 1979);
- up to 50% greater risk for all cancers in adulthood (Sandler, D.P., Everson, R.B., Wilcox, A.J.,& Browder, J.P.,1985); and
- a 50% greater risk for all cancers in childhood; 100% increased risk for non-Hodgkins lymphoma, acute lymphoblastic leukemia and Wilms' tumor (Stjernfeldt, M., Berglund, K., Lindsten, J., & Ludvigsson, J.,1986).

Because there are so many compounds in environmental tobacco smoke, it is difficult to determine the effect of any one chemical. But dozens of studies show adverse effects of ETS on fetuses and young children.

Pesticides

All pesticides are regulated by the Environmental Protection Agency. While registration files on individual pesticides generally include complete data on short-term health effects of pesticides, they are often missing data on long-term effects, such as mutagenicity, teratogenicity, and carcinogenicity. In fact, only 5 of some 425 active pesticide ingredients registered before 1984 have met the data requirements of current registration standards.

The example of the class of indoor pesticides known as organophosphates is instructive. Organophosphates are commonly used for roach, flea, and termite control in homes and schools. Originally, the German firm I.G. Farben developed these neurotoxins for use in Nazi gas chambers. After the war, the company sold the patents to American firms, which used the compounds as pesticides.

Organophosphates operate by inhibiting an enzyme that breaks down a neurotransmitter, causing nervous system dysfunction. In humans, symptoms of the dysfunction range from hyperactivity, restlessness and anxiety to convulsions, coma and death (Office of Technology Assessment, 1990).

Tests of the effects of organophosphates on animal fetuses and neonates have revealed changes in nervous system activity, including nerve transmission function, neuromuscular function, and brain electrical activity (Spyker, J.M. and Avery, D.L., 1977; and Rosenstein, L. and Chernoff, N., 1978). In another animal study, 15 of 16 organophosphates studied were found lethal at lower doses for young rats than for adult rats (Calabrese, E.J., 1986).

A recent study examined home concentrations of the organophosphate Dursban (Fenske, R.A., et al, 1990). Researchers looked at the breathing zone of a crawling infant and surface carpet residues, in ventilated and unventilated rooms, on the day of application of the pesticide and the day following. The results showed that an infant would be exposed to 1.2 to 5.2 the adult No Observable Effect Level (NOEL) of the pesticide. (NOEL is the dose below which no biological effects of a substance are seen.) The authors of the study state that there could be measurable toxicological responses in children and that "while such exposures are not likely to produce recognizable symptoms of (nervous system dysfunction), the possibility of cases with subclinical effects cannot be ruled out (p. 692)." They go on to say that because of accidents by minimally trained pesticide applicators and repeat sprayings for pests, the

exposure of infants to organophosphates used in ordinary circumstances is likely to be greater than that documented in the research situation.

Exposure to pesticides in the home has also been implicated in increased risk of cancer. A study by the National Cancer Institute (Lowengart, R.,Peters, J.M., Ciccini, Buckley, J., Bernstein, L., Preston-Martin, S., & Rappaport, E., 1987) found that children who were regularly exposed to pesticides at home, both indoors and out, were as much as seven times more likely than other children to have leukemia. A study on the incidence of brain tumors in children (Gold, E., Tonascia, J., Szklo, M., & Gordis, L., 1979) showed household pesticides to be one of the risk factors.

Volatile organic compounds

Volatile organic compounds (VOC) or solvents are liquids that turn to gas when exposed to air, making them easy to inhale. The National Institute of Occupational Safety and Health (NIOSH) has estimated that in 1984, 49 million tons of industrial organic solvents were produced in the United States. More than 500 different compounds or solvents have been identified in indoor air samples (Sheldon, L.S., Handy, R.W., Hartwell, T.D., Whitmore, R.W., Zelon, H.S., & Pellizari, E.D., 1988). VOC's are usually the chemicals implicated in the cases of sick building syndrome described earlier.

Many common household products contain VOC's. The VOC methylene chloride, which is both a carcinogen and a neurotoxin, is present in some brands of 12 household products: paint strippers, adhesive removers, spray shoe polish, adhesives and glues, paint thinners, glass frosting and artificial snow, water repellents, wood stains and varnishes, spray paint, cleaning fluids and degreasers, aerosol spray paint for automobiles, and automobile spray primers (Consumer Product Safety Commission, 1987). Calculations from an exposure survey conducted by EPA from 1981 1985 (Wallace, unpublished paper) indicate that methylene chloride levels in homes exceeded EPA's "negligible risk standard" of 1 in 1 million cancers by more than a factor of 10.

The solvent benzene is another VOC that is a proven carcinogen and neurotoxin. Many industrial plants emit large quantities of benzene, which is regulated as a hazardous substance under the Clean Air Act (OTA, 1990). However, 80% of human exposure to benzene is not regulated; it comes from tobacco smoke, wood stoves, home products, and hobbies, such as painting. Some researchers hypothesize that the increase in childhood leukemia rates from exposure to tobacco smoke is due to the presence of benzene in the smoke (Wallace, L.A., 1989).

The VOC vinyl chloride is a component of environmental tobacco smoke and some household degreasers. Carcinogenicity studies of young animals exposed to vinyl chloride show that the young are more susceptible than adults to cancer of the liver, mammary glands and skin. The incidence of liver cancer in exposed newborn rats was more than 40 times that in an adult population (Whyatt, R., 1989).

The volatile organic compounds in common household products come under little regulatory scrutiny. And no research has investigated whether low levels of exposure to these known neurotoxins can cause or exacerbate children's learning or behavior problems or mood shifts.

FLORENCE SHARP

Chemical sensitivity

Much of the current controversy over indoor air quality concerns the effect of exposure to very low levels of chemicals. Can a one part per million or one part per billion concentration of *anything* in the air really constitute a problem? (It is difficult to make such numbers meaningful: it helps to remember that one part per million corresponds to one cent in $10,000, and one part per billion to one cent in $10,000,000.) Regulation of indoor air quality is weak or nonexistent, and public awareness is slight. However, the segment of the population that is especially sensitive to chemicals has begun to raise public consciousness about the issues involved. Many researchers who believe that exposure to low levels of chemicals can be harmful emphasize the concept of "toxic load." They suggest that it is not the single exposure to a single substance that is harmful, but rather a multitude of daily exposures to toxins from air, water and food.

The controversy began with "sick building syndrome." Traditional toxicologists and medical researchers at first dismissed complaints of fatigue, headache, and respiratory problems occurring only in a particular place as psychosomatic. Today, after many years and documented incidents, sick building syndrome

is accepted as a real toxicological problem. Now the focus of controversy has shifted to individuals who have had an acute toxic exposure, as in sick building syndrome, and who subsequently continue to experience symptoms when exposed to very low levels of chemicals, typically volatile organic compounds.

The following vignette of one family's experience is excerpted from an account written and circulated by the family.

After months of construction, Jack, Miriam and their two children were finally going to be able to enjoy their house renovation. Everything was done, except for installing the carpet, and that was to occur the next day. The day arrived, the beige pile carpet was laid out, and, in no time, the family room was filled with furniture and cherished family possessions.

Within a few months, however, all members of the family were experiencing some off-and-on health problems—chapped and burning lips, headaches, unusual thirst, and burning sensations in the eyes, nose and sinus cavities. When the family were outside, their symptoms were alleviated; once the family came back inside, the symptoms reappeared after three or four hours.

Lower life forms were experiencing difficulties, too. Dead spiders sprinkled the carpet. Blackened tips appeared on the leaves of three new, healthy plants two days after purchase.

Over time, the family's health problems became more serious. They included symptoms of depression, incoherent thinking and speech, fatigue, and shortness of breath. Finally realizing that the symptoms might be related to the new room, the family called in an environmental specialist to examine the house. After extensive sampling, he concluded that the carpet had emitted an array of volatile organic compounds: ethylbenzene, formaldehyde, methacrylic acid, toluene, amines, and styrene. These substances had saturated the walls, floor and ceiling of the family room. The specialist recommended not only that the carpet be removed but also that all room surfaces be decontaminated before further occupation.

Once the rooms were scrubbed and their surfaces resealed, they became usable again. For the family, whose bodies had also been saturated, no such simple detoxification plan was available. Now, 10 years after the remodeling, family members still experience symptoms of mental confusion, fatigue, and eye irritation whenever they come into contact with perfumes, formaldehyde, pesticides, and most petrochemicals. Avoidance of these chemicals is their only protection.

This family's experience was unusual, but not unique. In 1990, the Consumer Product Safety Commission conducted a survey of more than 300 cases in which consumers had complained about upper respiratory symptoms, headaches, and fatigue associated with new carpeting. The Commission decided that the survey's limitations precluded any findings of cause and effect relationships between the gases emitted by new carpeting and health problems. It recommended that a more comprehensive, case controlled study be conducted to explore causality (Schachter, L., 1990). Similarly, in 1990 the National Federation of Federal Employees filed a petition requesting that emissions from new carpets be regulated by the EPA. (Some Scandinavian countries require that new buildings remain unoccupied until chemicals outgassed from building materials are at an acceptable level [Governor of Maryland's Task Force on Indoor Air Quality, 1989]). The EPA refused to issue regulations and instead asked carpet manufacturers to minimize any potential risk from carpet emissions voluntarily (*Indoor Air Review*, 1991) There is no evidence that the EPA considered the effect of carpet emissions on young children in making its decision.

Beginning research on chemical sensitivity

Controversy about chemical sensitivity will only be resolved by learning more about the physical symptoms that accompany exposure to chemicals. The National Academy of Science has recommended further studies in this field (Barinaga, 1991), and the EPA is sponsoring some research.

Two recent studies (Hudnell, H.K., Otto, D.A., House, D.E. & Molhave, L.,1990 and Koren, H.S., Devlin, R.B., House, D.E., Steingold, S. & Graham, D.E.,1990) focused on the change in upper respiratory passages from exposure to 22 volatile organic compounds that are commonly found in new or renovated buildings. The test subjects—healthy, non-smoking volunteers who had no known reaction to pollutants—spent 3 to 4 hours exposed to VOC's in a special steel chamber. The studies confirmed sensory irritation at a statistically significant level.

A third recent study compared physiological and psychological reactions of subjects who had reported experiencing sick building symptoms previous to testing with subjects who had never experienced symptoms. Exposure to VOC's resulted in statistically significant physiological changes in both groups which suggested that the ability to learn may be impaired by VOC exposure (Kjaergaard, S.,1990).

Government regulation

No single agency of the federal government has jurisdiction over indoor air quality. The Occupational Safety and Health Administration is responsible for worker safety, including air quality, but it concentrates most of its effort on industrial settings. The Environmental Protection Agency funds some indoor air quality research and serves in a coordinating capacity but does not have general authority. Other federal agencies are responsible for regulating specific pollutants or for protecting specific constituency groups.

Some substances that have been shown to be carcinogens, neurotoxins, or respiratory irritants are regulated within a certain context. For example, the ingredients in pesticides that actually kill pests ("active" ingredients) are regulated by the EPA. However, ingredients in pesticides that are not the pest killing agents ("inert" ingredients) are not regulated, even though they may be toxic to humans.

Major questions remain unresolved by current legislation: the amount of research needed, the incidence of illness that constitutes a health risk, and the trade-offs between health and economic benefits.

Toward a reasonable response

The reaction of the general public to information about indoor air quality (and toxins in general) ranges from jaded disdain to panic at each new report. And if it is hard for an adult to assess risks and make choices for himself or herself, it is almost infinitely more difficult to feel confident in making choices about indoor air quality that affect one's own young children or infants and toddlers in the community for whom one is to some degree responsible.

Certainly research findings point to the wisdom of extreme caution concerning the exposure of infants and toddlers to lead, radon, asbestos, environmental tobacco smoke, and pesticides in indoor air, even at low levels. Until further research is completed on the effects of other toxins, especially the volatile organic compounds, one can choose either to increase ventilation and minimize exposure, or to risk the consequences of taking no action pending further evidence of problems.

Individualized response patterns to toxic substances further complicate decisionmaking. Not every child exposed to mercury in latex paint becomes ill, nor did every guest at the Bellevue Stratford Hotel in Philadelphia develop the indoor air malady that came to be known as Legionnaire's Disease.

As in so many other circumstances, people who are knowledgeable and concerned about infants and toddlers must represent the special needs of this voiceless, voteless constituency. We must demand that new research on indoor air pollution consider the environments in which very young children spend their time and the special vulnerabilities of infants and toddlers. We must insist that new regulations of indoor air quality, from whatever source, be appropriate for this population.

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APHA/AAP Health and Safety Guidelines for Child Care

Supported by the U.S. Maternal and Child Health Bureau, the American Public Health Association (APHA) and the American Academy of Pediatrics (AAP) have been working for four years to develop health and safety performance standards for out-of-home child care programs. These will be issued in the forthcoming publication *Caring for Our Children - National Health and Safety Performance Standards: Guidelines for Out-of-home Child Care Programs*. The publication will address staffing, administration, program, facilities, nutrition, infectious diseases, and serving children with special needs. Specific topics include lead exposure, the quality of indoor lighting and ventilation, heating, cooling, water supplies, equipment maintenance, and toxic exposure. *Caring for Our Children* will make recommendations for licensing and community action related to health and safety in out-of-home child care programs.

For information about the publication, write to Marketing Department, American Public Health Association, 1015 15th Street, N.W., Washington, D.C. 20005.

Environmental Risks to the Infants and Toddlers of Migrant Farmworkers

by

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The agricultural workplace poses many threats to the health of migrant farmworkers and their children—accidents, heat, stress, fatigue, poor sanitation in the fields, lack of adequate water, sub-standard housing, poor nutrition, dust, and air-borne allergens. All of these factors play important, interrelated roles in affecting migrant workers' health and the health and development of their children. At the East Coast Migrant Head Start Project (ECMHSP), a non-profit, federally funded Head Start grantee, staff and participating parents are increasingly concerned about another potential hazard, the prevalent use of pesticides in farming. We see an urgent need for further documentation and research.

At the East Coast Migrant Head Start Project, staff and parents are concerned about possible relationships between pesticide exposure and the health and developmental problems of young children. We see an urgent need for further documentation and research.

ECMSHP serves approximately 5,000 children each year. Infants and toddlers make up two-thirds of this group, as well as a large proportion of ECMHSP's waiting list. As in all Head Start programs, approximately 10 percent of the children served by ECMHSP have diagnosed disabilities. But the monthly logs from each center that document the kinds of disabilities diagnosed or suspected in the children served suggest that the health problems and disabilities of the children in our program tend to be more severe than those experienced by children in other Head Start programs.

What families tell us

What do migrant farmworker families tell us about their experiences with pesticides? Many mothers describe working in fields where pesticides had been sprayed while they were pregnant, and they believe that pesticide exposure is related to their children's chronic health problems and disabilities. Other mothers, often with similar experiences, do not report pesticide exposure when talking about their chidlren's health history. Some parents make a connection between pesticide exposure and a child's illness or disability years after the child's death. Here are some of the stories we have heard.

Yesenia A., who has a genetic disorder known as Marfan's Syndrome, has developed chronic blepharitis (frequent tumors on her eyelids). Her physician has said that this condition was a reaction to chemical exposure. (Yesenia's health history is one of the few

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that includes such specific information.) Yesenia's mother was taking her to the fields on weekends. There were no washing facilities in the fields. The child would rub her eyes. Every time Yesenia goes to the fields, tumors develop on her eyelids within several days of exposure.

Mayra C.'s mother told us that she worked in fields sprayed with pesticides and went into labor in the fields. Mayra has a congenital defect of the esophagus, chronic otitis media, and related communication problems.

Yolanda is Mayra's cousin. She also has respiratory problems, chronic otitis media, and related communication problems. The two sisters, mothers of Mayra and Yolanda, travel together as migrant farmworkers, but Yolanda's mother has never reported exposure to pesticides to health workers or ECMHSP staff.

Ana, Juana and **Luis** are triplets, born at 25 weeks gestation to a mother who worked in fields sprayed with pesticides. Ana weighed 2.7 pounds at birth; Juana, three pounds, and Luis 3.2 pounds. They all had respiratory distress syndrome and remained in the hospital for two months before coming home. Although the health of Juana and Luis has improved, Ana has developed right hemiparesis and global developmental delays.

Vanessa R., eight years old, is in a public school program for gifted children. She shows no sign of severe health or developmental problems, Vanessa, however, was born with spina bifida and failed to thrive as an infant. Her mother was the first independent woman crew leader in southwest Florida; she spent day and night in the fields at a time when growers were using strong pesticides. Vanessa was hospitalized repeatedly to repair the spina bifida and nearly died several times.

Rosa M. is an outreach worker in an ECMSHP project that is specially funded to serve migrant children with disabilities in two areas of Florida that have no other migrant Head Start centers. Rosa now tells of working in the fields during one of her pregnancies and remembers being sprayed with pesticides. Spray got on string she was using to tie vines. She remembers her eyes burning and her lungs hurting. Rosa gave birth to a full-term girl whose intestines were outside her body at birth. The baby had seizure and feeding disorders and was eventually diagnosed with Cornelia de Lange Syndrome (although she did not have every feature of the syndrome). The baby died at 14 months. Rosa never reported her pesticide exposure in her child's health history, but she has begun to wonder about a connection.

DAVID KREIDER

What we need to learn

We are alarmed and concerned about what program families tell us about their experiences with pesticides. Even if the pesticides to which they and their children are exposed are minimally harmful in themselves, the working and living conditions they endure are an outrage—lack of water, soap and toilet facilities in the fields; housing exposed to pesticides carried by wind drift.

We know that there are difficulties in documenting, tracking, and analyzing the effects of pesticide exposure in migrant farmworker families. This is by definition a mobile population. It is difficult to determine which pesticides were used and when; the effects of different mixtures of pesticides; the wide range of pesticides to which any one worker was exposed in a given season; and the impact of factors apart from pesticide exposure that can have an adverse effect on health. Workers' failure to document and report pesticide exposure and illness and a lack of adequate records and record keeping procedures in the farm industry and among programs serving this population also contribute to the difficulty in determining precisely the long-term effects of pesticide exposure.

But the challenge is not insuperable. There are many sources of data, including information collected through the Migrant Student Record Transfer System and child and family data collected by Migrant Head Start Programs, which could be compared with data on other Head Start families as well as the general population. A comprehensive survey of 400 migrant worker families that is now underway may prove revealing.

Meanwhile we will work to raise the awareness of families, educators, health, and diagnostic practitioners concerning the potential hazards of pesticide exposure. We will continue to ask questions. We will continue to tell growers—and legislators—about what families, center staff, and community service providers see as they care for young children exposed to pesticides. We will continue to bear witness to our own experiences and those of our families. We continue to speak out.

CASSP/NIMH Support for Research Demonstration Projects

Beginning in fiscal 1991, the National Institute for Mental Health (NIMH) has issued a standing program announcement which supports research demonstration projects designed to improve the delivery of mental health services to children, adolescents and their families. Although demonstration projects funded in the past by NIMH's Child and Adolescent Service System Program (CASSP) did not always emphasize research and evaluation, it is now considered essential, according to the announcement, that research and evaluation be an integral part of every demonstration project funded by the institute. Research demonstration projects require collaboration between researchers and state program staff in project design, application preparation, and project implementation. State mental health authorities must make the actual applications for CASSP grants.

For information on developing a CASSP/NIMH research demonstration project, contact: Judith Katz-Leavy, Chief, Technical Assistance Program, Child and Family Support Branch, Division of Applied and Services Research, National Institute of Mental Health, 5600 Fishers Lane, Room 11 C-05, Rockville, MD 20857, tel: (301) 443 1333.